

Frontal lobe dysfunction in cerebrovascular disease

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Summary

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The frontal lobe is the largest lobe of the brain and it is thus commonly involved in stroke. Moreover, almost one in five strokes is limited to the prerolandic areas. This high frequency of anatomical involvement is in sharp contrast with the apparent rarity of clinical frontal dysfunction in stroke. It is remarkable that frontal behavioural syndromes have rather uncommonly been reported in patients with stroke as compared to patients with other diseases, such as brain tumour. This fact is paradoxical, because an acute process (stroke) is expected to yield more clinical dysfunction than a more chronic disease (tumour). A volume effect may be the main factor leading to this phenomenon.

Another interesting aspect of frontal strokes is the contribution of so-called “silent” strokes, the recurrence of which may nevertheless lead to intellectual decline and compromise recovery from another stroke with more specific neurologic dysfunction. The contribution of stroke to understanding of frontal lobe dysfunction is important, because of the focal nature of this disease, and a great opportunity for clinical-topographic classification correlations.

One of the first modern attempts to develop a clinical-topographic classification of frontal lobe lesions came from the school of Luria, who tried to delineate three main types of frontal lobe syndromes (premotor syndrome, prefrontal syndrome, medial-frontal syndrome).

Recent anatomic correlates using MRI make it possible to improve this classification. We suggest considering six main clinical-anatomic frontal stroke syndromes: (1) prefrontal, (2) premotor, (3) superior medial, (4) orbital-medial, (5) basal forebrain, (6) white matter.

Finally, another fascinating topic relates to frontal lobe symptomatology due to stroke sparing the frontal cortex or white matter. This occurs mainly in three instances: lenticulo-capsular stroke, caudate stroke and thalamic stroke. Studies using blood-flow or metabolism measurements suggest that diaschisis (frontal lobe dysfunction from a remote lesion) may play a role. We believe that this is more likely to be related to dynamic interruption of complex circuitry than to static frontal lobe deactivation.

Keywords: stroke; frontal lobe; behavioural neurology

Introduction

The frontal lobe is the largest globe of the brain and it is logical that it should often be involved in stroke. This high frequency of anatomical involvement is in contrast with the apparent rarity of clinical frontal lobe dysfunction in stroke. The reason for this discrepancy is not clear, but it may have to do with the site and size of lesions, as it is uncommon to have a very large cerebral volume involved in stroke in conscious patients, or the lesion is not limited to the frontal region so that many other manifestations will mask frontal lobe dysfunction.

While it is admitted that the frontal lobes play a critical role in the mental processes, both attempts at systematising their functions and at making a clinical-topographic classification remain more incomplete and speculative than for any other part of the brain.

Most of the more precise modern correlations in focal lesions of the frontal lobe have been derived from the work of A. R. Luria, who emphasised three main syndromes of frontal lobe dysfunction: (1) *premotor or postero-frontal syndrome*

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(Brodmann's area 6, 8, 44, 45), corresponding to deautomatisation of motor acts and revival of elementary automatisms, with difficulty in performing smooth movements composed of a chain of successive links (loss of smooth switch, disturbance of "kinetic melodies"); (2) *prefrontal syndrome* (Brodmann's area 9, 10, 46), corresponding to impaired regulation of voluntary actions and programs, and loss of self-criticism; (3) *medial frontal syndrome* (Brodmann's area 11, 12, 24, 25, 33, 47), corresponding to affective disinhibition, impulsiveness, distractibility and marked loss of the selectivity of mental process (pseudo-confused/pseudo-psychiatric aspect).

Recent advances in clinical-anatomical correlation secondary to the dramatic improvement of neuro-imaging during the last decade have allowed to refine it. I would like to propose the following approach to the frontal lobe dysfunction associated with stroke:

- 1 *frontal lobe symptomatology due to frontal stroke*
 - 1.1 dorsolateral syndromes (middle central cerebral artery territory)
 - 1.1.1 prefrontal syndrome
 - 1.1.2 precentral (premotor) syndrome
 - 1.2 mediofrontal syndromes (anterior cerebral artery territory)
 - 1.2.1 superior medial syndrome
 - 1.2.2 orbital-medial syndrome
 - 1.3 basal forebrain syndrome (anterior communicating artery territory)
 - 1.4 frontal white-matter stroke
- 2 *frontal lobe symptomatology due to other strokes*
 - 2.1 lenticulo-capsular stroke
 - 2.2 caudate stroke
 - 2.3 thalamic stroke

Finally, over 15% of the patients with first-ever stroke have one or more asymptomatic infarcts on CT, which preferentially involve the frontal lobes. Though these lesions are called "silent", they may play an important clinical role in case of recurrences and contribute to the development of progressive or stepwise dementia with frontal lobe dysfunction.

Dorsolateral syndromes

The prefrontal syndromes remain perhaps the less well known of the frontal stroke syndromes, despite the large volume of brain parenchyma which can be involved here. In stroke, this syndrome is due to involvement of the branches of the prefrontal artery or the precentral artery

from the middle cerebral artery (MCA). It is much more common to see a partial clinical picture, with emphasis on a particular type of frontal dysfunction.

Prefrontal syndromes

Loss of programming activities. This is often considered as "the" prototype of clinical dysfunction associated with prefrontal damage, but this picture has poorly been reported and evaluated in stroke patients.

Utilisation and imitation behaviours. Compulsive manipulation of tools has been described by Mori and Yamadori (1982) in anterior cerebral artery (ACA) territory infarction. Later, Lhermitte (1983) developed the concept of utilisation behaviour, mainly with lesions involving the late parts of the prefrontal areas. Imitation behaviour represents the first stage of the "environmental dependency syndrome". Imitation/utilisation behaviour has not been studied extensively in stroke patients. Our experience suggests that they are commonly overlooked in routine situations.

Grasp reflex. A grasp reflex may occur in over 5% of hemispheric strokes, more often with multiple lesions. The main locus involves the frontal lobe, less often the deep nuclei or white matter and never a post-rolandic area. Within the frontal lobe the dorsolateral and premotor regions are involved in only ¼ of cases, while fronto-medial lesions are more common.

Perseverations. Three main types of perseverations have been delineated by Sandson and Albert (1984): (1) *recurrent* perseveration corresponds to the repetition of previous response to subsequent stimulation; (2) *stuck-in-set* perseveration is an inappropriate maintenance of a category of activity; (3) *continuous* perseveration corresponds to abnormal prolongation of a current activity without interruption. They may coexist and precise anatomic correlates are lacking. The first two types have mainly been associated with prefrontal lesions, usually involving the dorsolateral region, while the third type is more suggestive of deep hemispheric/basal ganglia damage.

Response-to-next-patient stimulation. In 1988 we reported this behaviour in 11 (8%) of 134 patients with right-sided hemispheric strokes (Bogousslavsky and Regli, 1988). The patients responded to stimuli directed at other patients as if the stimuli were directed at them. This repetition of a particular behaviour was interpreted as a form of perseveration (stuck-in-set). It occurred only in severe strokes involving the right prefrontal

Table 1

lesion	aphasia or other speech disturbance
frontal operculum (so-called "Broca's area")	transient decrease of speech output (NOT Broca's aphasia)
frontal operculum + underlying white matter	transcortical motor aphasia
lower motor strip	restricted arthric disturbances (phonetic disintegration of Roch Lecours and Lhermitte)
lower motor strip + underlying white matter, including preentricular white matter	same + phonemic substitutions and writing impairment
all the above (with involvement of the limbic-periventricular fibres of the medial subcallosal fasciculus)	classical Broca's aphasia

region, usually with parietal and temporal extension. Associated disturbances such as hemiparesis, hemineglect, anosognosia and motor imperistence were usual.

Delusions. Delusions involving orientation in time and place, past events or identities of familiar individuals have been reported in dorsolateral prefrontal strokes, more often right-sided than left-sided. *Reduplicative paramnesia* of Pick has also been reported in prefrontal stroke. It corresponds to the belief that people, places or events have been doubled. It should be differentiated from *misidentification syndromes*, where the original (usually a person) is believed to have been replaced by an impostor (while, in reduplicative paramnesia, duplication does not imply that one of the two is fake). Classical misidentification syndromes include Capgras syndrome or "illusion des sosies" (original replaced by impostor, i.e. a hypo-identification phenomenon), Frégoli syndrome (psychic characteristics of person are found in other persons, i.e. a hyper-identification phenomenon), intermetamorphosis syndrome (same as preceding but also with physical characteristics) and subjective doubles syndrome (same as preceding, but involving the subject's own characteristics). In fact, it is probably an exception for these syndromes to develop in stroke patients who do not have a preexisting psychiatric disorder.

Premotor syndrome

Loss of kinetic melody (limb kinetic apraxia). Luria developed this concept to explain the difficulty that patients have to switch smoothly from one motor act to another, while each of these acts can be performed normally in isolation. This inability is manifested by motor perseverations, interruption of motor sequences and decomposition of acts into elementary motor patterns. This disturbance is common in stroke involving the precentral artery territory but it is typically

overlooked because routine neurologic examination fails to detect it.

Motor weakness. Freund and Hummelsheim (1985) reported 9 patients with infarct involving the premotor cortex but sparing the precentral area, who had limb kinetic apraxia, but also contralateral moderate weakness of shoulder and hip. Elevation and abduction were mainly affected, in contrast with proximal weakness secondary to precentral lesion, in which abduction, adduction, elevation, flexion and extension are involved to the same extent (and without kinetic apraxia).

Hemineglect. The concept of hemineglect from frontal lesion (mainly on the right) was initiated by Castaigne et al. and Heilman et al. in 1972, who emphasised its motor components. However, as a rule, neglect is more often associated with retrorolandic than prerolandic stroke, and motor neglect may even be found with parietal lesion in the absence of marked sensory neglect. The term "directional hypokinesia" may be appropriate to describe the main feature of neglect from premotor lesion (also from subcortical lesion), in which prolonged reaction times are registered for movements directed contralaterally.

Motor imperistence. This inability to maintain a motor act or a posture for more than a few seconds is probably related to an impairment of the mechanism of directed attention necessary to sustain motor activity. This dysfunction has been reported with premotor stroke, usually on the right. It may be bilateral or mainly contralateral. A clinical form is the *inability to maintain eye closure* (<10"), an extreme of which may be complete impossibility to initiate eyelid closure. Ghika et al. (1988) showed that inability to close the eyes on command (with preservation of automatic/synkinetic movements) corresponds to frontal damage interrupting the connections between the supplementary motor area and the premotor region.

Motor persistence. Abnormal maintenance of a particular, often uncomfortable position has also

been called "catalepsy" in a psychiatric setting. In our experience with strokes, it is associated with frontal damage involving the premotor and immediately anterior prefrontal region, on the right side. This phenomenon has poorly been studied, though it is not uncommon in the acute course of stroke.

Aphasia. "Motor aphasia" from left frontal damage has been mentioned since historical reports on aphasia (aphemia or pure apraxia of speech [Broca, 1861; Bastion, 1878], pure anarthria [Marie, 1906], pure word dumbness [Wernicke, 1906], pure motor aphasia [Dejerine, 1914]). More recently, Alexander et al. (1990) proposed an interesting clinical-topographic classification of aphasia from frontal lesions (table 1).

Global aphasia without hemiparesis is usually suggestive of double (anterior + posterior) lesion, but it may also develop from a single frontal infarct (involving F₂ and F₃), usually with good recovery.

Mediofrontal syndromes

Superior medial syndrome

In stroke, this syndrome is typically associated with ACA infarction, which is present in 1–4/200 patients with cerebral infarct. Involvement of the supplementary motor area (SMA), its surroundings and the corpus callosum seems the major determinant for this clinical syndrome.

Response preparation and inhibition. A lateralised finding has been reported by Verfaellie and Heilman (1987), who noted that patients with left-sided lesion could well inhibit unrelated acts and prepare a task, while patients with right-sided could not. They suggested that the right SMA was dominant for response preparation and inhibition, possibly by modulating levels of excitability of motor system.

Compulsive manipulation of tools. In 1982, Mori and Yamadori reported a patient with infarct involving the left mesial frontal lobe and anterior corpus callosum who showed compulsive grabbing and use of objects with the right hand, while the left hand tried to restrain the right hand. This report corresponds to the first detailed description of utilisation behaviour.

"Callosal" disturbances. They are often overlooked. In fact, up to ¼ of the patients with ACA infarct may show *unilateral left ideomotor apraxia*; involvement of the anterior ¼ of the corpus callosum was present in all patients. *Unilateral left agraphia* or tactile anomia has also been reported after left pericallosal artery territory infarct. *Alien*

hand corresponds to apparently purposeful movements that appear to be dissociated from conscious volition. *Loss of bimanual coordination* with intermanual conflict (diagonistic dyspraxia) probably represents an extreme form of alien hand in large ACA territory infarct.

Grasp. Nearly ¾ of the cases are secondary to medial-frontal lesion, with systematic involvement of the cingulate gyrus, less often the SMA. Inter-hemispheric differences have not been found. Usually *both* hands are involved, and the patient can partly inhibit the grasp. "*Grasp reaction*" (*groping*) is closer to utilisation behaviour and more uncommon than grasp reflex. *Foot grasping* has been reported but we were unable to find a single case in 27 patients with ACA infarct.

Aphasia. Transcortical motor aphasia has been emphasised in left ACA stroke involving the supplementary motor area or underlying white matter. Deep extension has been linked to poor articulation, while anterior capsular-caudate involvement is associated with comprehension impairment. We found that posterior extension toward supplementary sensory area was not associated with comprehension disturbances.

Graphomania (graphorrhoea). This type of hypergraphia with semantic incoherence has been reported in superior-medial frontal tumour, but I am not aware of a vascular case. Graphomania should be differentiated from Yamadori's hypergraphia from right hemisphere damage (no semantic incoherence) and from echographia (triggering by an external stimulus).

Hemineglect. Although marked hemineglect seems very uncommon, unidirectional hypometria and hypobradikinesia have been reported.

Other. Other disturbances include left-handed mirror writing, facial palsy with inverse automatic-voluntary dissociation, apraxia, dyscalculia, inverted vision and urination behaviour.

Bilateral ACA infarcts

The clinical picture may associate combinations of the above-mentioned disturbances, but two suggestive pictures must be mentioned:

- 1 complex dysfunction with indifference, docility, inappropriate urination and lack of attention, combined with confabulatory syndrome due to damage to bilateral anterior cingulate gyrus, adjacent white matter and fornices;
- 2 akinetic mutism or its variants are the usual neurologic picture with more extensive lesion.

Orbital medial syndrome

The fascinating behavioural disturbances linked to orbital-medial frontal damage have been little studied, probably because of the nature itself of these disturbances. The best-documented case is non-vascular (patient EVR) (Eslinger and Damasio, 1985), in whom there was surgical damage to the orbital-medial frontal region on both sides, with sparing of the superior-medial region and basal forebrain. The patient had normal standard neuropsychological examination and “measurable intelligence”, but showed abnormal behaviour in that he was unable to use planned patterns in real-life situations, which failed to evoke the normal response patterns. This sort of strategy application disorder was in sharp contrast with “normal” neuropsychological functions.

This clinical picture has not been studied in stroke. Grasp or alien hand have been reported with infarct in this location, but usually in association with extension toward the more superior part of the medial frontal region.

Basal forebrain syndrome

Infarction in the anterior communicating artery (ACoA) territory may encompass the septal area, nucleus accumbens, nucleus of the diagonal band of Broca, paraterminal gyrus and nearby tracts, often with extension toward the anterior limb of internal capsule, hypothalamus and globus pallidus. The usual cause of infarct is ACoA aneurysm with subarachnoid haemorrhage. The clinical picture is dominated by amnesia, which is often severe (Damasio et al., 1985). A Korsakoff-like picture was first reported by French authors (Brion et al., 1968), who emphasised “fabrications” by the patients, such as “extravagant or fantastic paraphasias”, which, in association with the use of emphatic and affected terms and impossibility of brief responses, gave the aspect of an incoherent but fluent speech. Damasio et al. (1985) described a multimodal amnesic syndrome close to Korsakoff’s syndrome, but with more extensive and dense memory impairment; anterograde amnesia involved generic/semantic as well as contextual/episodic components, while retrograde amnesia was severe, involving all decades of life and sparing only semantic/generic material. There was a good effort of cuing. “Wild” fabrications were also emphasised, being less close to previous life than in classical Korsakoff’s syndrome. Damasio et al. hypothesised that secondary hippocampal dysfunction was responsible for amnesia, because of

the strong connections between basal forebrain and hippocampus.

Frontal white-matter strokes

The frontal white matter is large and it is often involved in anterior borderzone infarction, medullary artery (superficial perforators) ischaemia and “vascular dementia”, including Binswanger’s disease. However, little is known about strokes limited to the frontal white matter, as only scattered and heterogeneous cases have been reported. Clinically, hemineglect is uncommon if the cortex or basal ganglion are not involved. Grasp and cortical disinhibition reflexes may occur, but are less common than with cortical damage. Transcortical motor aphasia may be a common consequence of left-sided injury, in relation to medial subcallosal fasciculus and surrounding white-matter damage. Bilateral damage can lead to “the syndrome of loss of psychic self-activation” without coexisting amnesia, but often with stereotyped mental activity.

Frontal symptomatology from stroke outside the frontal lobe

Lenticular-capsular stroke

Neurobehavioural changes including clinical frontal dysfunction secondary to subcortical strokes have been reported and studied well. Although the mechanisms may be complex, these changes have usually been attributed to disconnection of cortical structures from the basal ganglia and related subcortical structures. The concept of an interruption of functional circuitry may be more appropriate, as disconnection overemphasises a function localised within the cortex.

Three major clinical pictures can be isolated, the first with unilateral infarct, the other two with bilateral or multiple strokes.

1 Lower capsular genu syndrome. It corresponds to infarct localised in the territory of direct perforators from the carotid apex. Akiyuchi et al. (1990) were the first to delineate this syndrome, before Tatemichi et al. (1992) reported a detailed study. Patients develop acute-onset lethargia and abulia often with confusion and memory loss justifying the term “single-infarct” or “strategic-infarct” dementia.

2 Bilateral pallidal-striate stroke. When the internal capsule is spared, the picture is dominated by neurobehavioural changes, with apathy and lack

of motivation but without real depression. The extreme form associated severe loss of drive, affectivity and interest, which contrasts with the possibility of appropriate activation by external stimuli and has been called psychic akinesia or athymhormia (Laplane et al., 1984; Habib and Poncet, 1988). One characteristic is the total or relative lack of impairment on standard neuropsychological testing, provided that the patient is adequately awakened/aroused and stimulated by the examiner.

3 Multiple lacunar infarcts. Several authors have emphasised impairment of frontal systems as the main neurobehavioural dysfunction associated with bilateral multiple lenticulostriate lacunar infarcts, providing a continuum with the controversial concept of subcortical dementia of vascular origin.

Caudate stroke

In *unilateral* caudate head stroke, loss of drive and apathy similar to what can be found in lenticular stroke has been reported to coexist with marked obsessive stereotypia and checking rituals, sometimes with affective disinhibition bursts, so that misdiagnosis as psychiatric illness is a real danger (Pedrazzi et al., 1990). Standard neuropsychological examination may be unimpaired.

In *bilateral* involvement, affective behavioural changes may be prominent, with vulgarity, impulsiveness, violent outbursts, hypersexuality and sometimes minor criminal behaviour, contrasting with coexisting indifference, loss of independence and decreased self-care.

As a functional correlate, marked frontal lobe hypoperfusion on the side of the caudate lesion has regularly been reported in association with these behavioural changes (Pedrazzi et al., 1990), perhaps by interrupting the limbic loop of the striatopallidal complex. The caudate has two main frontal lobe connections, the first with the dorsal-lateral region, the second with the lateral orbital cortex, which may explain in part the apparently contradictory coexistence of disinhibition and inhibition behaviours.

Thalamic stroke

It is interesting that a full picture of "frontal dysfunction" has not often been reported in thalamic stroke, though some frontal-limbic behavioural changes can develop after tubero-thalamic or paramedian thalamic damage. Only a few reports

have emphasised the possibility of "frontal lobe dysfunction" as the leading clinical disturbance in thalamic stroke, with impaired complex executive behaviours and decreased response inhibition, insight and judgement. Eslinger et al. (1991) reported utilisation behaviour as "hyperinstrumental" (i.e. coherent and directed toward the object function). A manic-like disorder with inappropriate laughs, jokes and comments, delirium and extraordinary confabulations has also been reported with right paramedian infarct (Bogousslavsky et al., 1988).

Isolated "loss of psychic self-activation" has been found with bilateral dorsomedial nucleus infarct (Bogousslavsky et al., 1991). The association of amnesia, apathy and decreased awareness forms the core of what has been called "thalamic dementia".

Clinical frontal dysfunction may correspond to damage to the limbic loop of the striatopallidal complex. Cerebral blood-flow studies have emphasised frontal hypoperfusion as a correlate of frontal symptoms in thalamic stroke.

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